

	CKMB-	CKMB+	TnT > 0.2	TnT < 0.1	TnT 0.1-0.2
Event Rate	27.4%	37.9%	38.7%	25.1%	62.8%
p value*	NA	-0.05	-0.001	NS	-0.0001

* Compared to CK negative group

Surprisingly, the additional risk with high TnT levels persisted even within the "high normal" range (0.05-0.1) with a 38.3% event rate.

Conclusions: TnT is a powerful predictor of late adverse events in suspected coronary syndromes. The greatest risk occurs in patients with intermediate levels, probably signifying an incomplete event. Thus, TnT levels between 0.05 and 0.2 identify a cohort who would have "ruled out" for acute MI by traditional standards, but remain at high risk for late events. The ability to risk-stratify this subgroup has significant implications for early and aggressive treatment strategies.

1083-135 Relationship of Anterior ST Depressions to Posterior Injury Pattern During Acute Coronary Artery Occlusions

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Anterior ST depressions (Ant. ST dep.) during acute coronary syndrome may represent either ant. subendocardial injury (Inj.) or posterior (post.) wall myocardial infarction (MI). To understand the mechanism of ant. ST dep., we recorded 15 lead ECGs (routine 12-leads with 3 simultaneous post. chest leads V7 to V9) during single vessel PTCA. Patients (pts.) with LBBB, recent MI, previous MI in same location, and use of perfusion balloon were excluded.

Results: Of the total of 224 pts. studied, 138 pts. showed Inj. pattern in any myocardial territory, while the 86 pts. showed ant. ST dep. Data are presented as n (percent). Cx = circumflex artery.

	Cx n = 75	RCA n = 77	LAD n = 72	Total n = 224
Ant. ST dep.	31 (41)	27 (35)	4 (5)	62 (28)
- with Post. Inj.	14 (18)	1 (1)	0 (0)	15 (7)
- with Int. Inj.	4 (5)	17 (22)	0 (0)	21 (9)
- with no Inj.	13 (17)	9 (12)	4 (5)	26 (12)
All Post. Inj.	36 (48)	1 (1)	1 (1)	39 (17)
All Inj. in 15 leads	42 (56)	43 (56)	53 (74)	138 (62)

While Post. Inj. pattern was seen mostly in Cx occlusions, ant. ST dep. was seen almost equally in Cx and RCA occlusions (41% vs. 35%). Of the 38 patients with Post. Inj. pattern, only 15 showed ant. ST dep. (sensitivity of 39%). Of the 62 patients with ant. ST dep., only 15 showed post. ST elevations (predictive value 24%). Of the 186 patients without post. ST elevation, ant. ST dep. was absent in 162 pts. (specificity 87%). Of the 29 pts. (26 with no injury in above table with 3 isolated Post. Inj.) without any injury pattern on 12 lead ECG, only 13 pts. (45%) were due to circumflex occlusions.

Conclusion: Ant. ST dep. during acute coronary occlusion is not sensitive for inferior or posterior Inj. pattern, nor does it predict the culprit vessel.

1083-136 Troponin I Elevations in the Absence of an Ischemic ECG Predicts Cardiac Events in Patients With Chest Pain

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Troponin I (TnI) has been shown to be a sensitive marker of myocardial necrosis and predictor of adverse outcomes. However, its diagnostic and prognostic capabilities in patients (pts) without ischemic ECGs have not been reported.

Methods: Serial sampling of TnI, CK and CK-MB was performed over 8 hours in 1338 consecutive pts without ST elevation or depression who were admitted for possible MI. MI was defined as an elevation in CK-MB of ≥ 8 ng/dl with a relative index (CK-MB \times 100/CK total) ≥ 4.0 . End-points were death (D), MI and revascularization (REV).

Results: MI (n = 156) or death (n = 12) occurred in 161 pts (13%), and an additional 158 (11%) underwent revascularization (REV). TnI ≥ 1.0 ng/ml (n = 227) was more sensitive than TnI ≥ 2.0 ng/ml (n = 160), but less specific. Relative risks (RR), Sensitivity (SN) and specificity (SP) and 95% confidence intervals for identifying pts with cardiac events are shown below.

	MI or D	MI, D or REV
TnI ≥ 1.0	6.7 (3.3 to 12)	5.7 (3.7 to 8.0)
TnI ≥ 2.0	5.3 (3.2 to 7.8)	5.4 (3.1 to 8.4)
SN TnI ≥ 1.0	88% (82-92)*	52% (47-58)*
SN TnI ≥ 2.0	75% (68-81)	41% (36-47)
SP TnI ≥ 1.0	94% (92-95)*	95% (93-96)*
SP TnI ≥ 2.0	97% (96-98)	97% (96-98)

* p < 0.01 TnI ≥ 1.0 compared to ≥ 2.0

Conclusions: Elevations of TnI in pts admitted for possible myocardial ischemia identify those with an increased risk for cardiac events. Lowering the threshold for an abnormal TnI improves sensitivity with minimal loss of specificity.

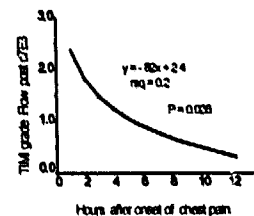
1084 Antiplatelet Therapies in Acute Myocardial Infarction

Tuesday, March 31, 1998, 9:00 a.m.-11:00 a.m.
Georgia World Congress Center, West Exhibit Hall Level
Presentation Hour: 10:00 a.m.-11:00 a.m.

1084-149 Predictors of Coronary Reflow With Intravenous Chimeric 7E3 Antibody in Acute Myocardial Infarction

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We have recently shown that intravenous chimeric 7E3 antibody (c7E3) can reverse coronary occlusion in patients with acute myocardial infarction (AMI) in the absence of exogenous plasminogen activator. However, some patients demonstrate persistent occlusion. The goal of this study was to elucidate clinical predictors of restoration of coronary flow with c7E3. Twenty four patients presenting with AMI underwent control angiography followed by c7E3 administration. Repeat angiography 10 minutes later revealed improvement in TIMI grade flow in 15/24 (62%) patients (Group 1) and no change in TIMI flow in 9/24 (Group 2). Mean TIMI grade flow after c7E3 was 2.0 ± 0.65 in Group 1 vs. 0.1 ± 0.3 in Group 2, $P < 0.001$. Univariate predictors of reflow included female gender ($P = 0.053$), absence of multivessel disease ($P = 0.02$), absence of smoking ($P = 0.07$) and shorter duration from onset of chest pain to c7E3 administration ($P = 0.028$). Logistic regression analysis identified chest pain duration and smoking as independent predictors of reflow post c7E3 with an odds ratio of 0.14 and 0.63 ($P = 0.055$ and 0.028) respectively.



Conclusion: 1) The frequency of coronary reflow with c7E3 in AMI is a time dependent process with highest reflow rate in infarcts less than 4 hours old. 2) Occlusive thrombus in smokers is more resistant to dissolution by c7E3 antibody.

1084-150 Pre-hospital Treatment With Abciximab in Acute Myocardial Infarction With Direct Percutaneous Transluminal Coronary Angioplasty

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Recent studies emphasized the benefit of Abciximab (Ab) in acute coronary syndrome angioplasty. We test the hypothesis that early (pre-hospital) treatment with Ab can affect infarct related artery (IRA) patency and improve the results of direct PTCA in AMI. In a single center with usual practice of direct PTCA in AMI, 23 patients, 16 males gender (62 ± 13 y) were included in a pilot study with pre-hospital treatment by Ab. Drug regimen included pre-hospital treatment with aspirin, bolus of heparin (70 U/kg), nitrates, β -blockers, bolus of Ab 0.25 mg/kg followed by an infusion of 0.125 mcg/kg/min. Time between Ab bolus and the onset of the symptom was 213 ± 188 mn, and coronary angiogram was performed 35 ± 17 mn later, in 10 inferior, 4 lateral, and 9 anterior AMI (EF $63 \pm 13\%$). TIMI flow in IRA (8 LAD, 1 Diagonal, 4 CX, 10 RCA) assessed by 2 independent observers was: TIMI 0 = 10 (43%), I = 2: II = 4, III = 7 (30%). After PTCA (22/23) and stent implantation (22/22), all pts have TIMI III flow in IRA. In hospital outcome was: no death, CABG, MI, or acute closure, 1 transfusion, 3 hematoma, 1 aneurysm, no surgical repair. After a mean follow-up of 2 months, we noted 1 MACE with 1 in stent acute closure (2 months) treated with Ab and new PTCA, and 4 pts (17%) expected cutaneous allergy related to ticlopidine. In this pilot study, pre-hospital treatment by Ab in AMI is safe, without serious adverse event, and Ab seems to improve TIMI flow in IRA, and facilitate PTCA. Larger series has to confirm these results.